4.1 Environmental Pollution and Disease: Links Between Exposure and Health Outcomes

Many studies have demonstrated an association between environmental exposure and certain diseases or other health problems. Examples include radon and lung cancer; arsenic and cancer in several organs; lead and nervous system disorders; disease-causing bacteria such as *E. coli* O157:H7 (e.g., in contaminated meat and water) and gastrointestinal illness and death; and particulate matter and aggravation of cardiovascular and respiratory diseases.

As mentioned in Section 4.0, indicators of outcome and exposure can be important tools both for elucidating these links and monitoring the success of environmental management efforts. Indicators are one of several components needed to establish linkage. Other important components include ambient pollutant measures and toxicological, epidemiological, and clinical studies. Three case studies are described in this section to demonstrate how indicators can be used to establish associations between exposure and effect and to evaluate environmental management actions.

Case Study on Waterborne Disease

This case study focuses on the impact of drinking water treatment on the decrease in mortality related to waterborne diseases. It demonstrates the valuable contribution to public health protection that can occur when the link between exposure and health outcomes is successfully made. As the case study describes, officials knew there was a high incidence of gastrointestinal disease, but they were not able to protect human health until they understood what caused these diseases. Based on this connection, officials were able to take effective action to protect public health. They also were able to use an outcome measure (deaths due to typhoid) to evaluate the success of these protective actions.

At the beginning of the 20th century, waterborne diseases such as typhoid fever and cholera were major health threats across the U.S. More than 150 in every 100,000 people died from typhoid fever each year. Deaths due to diarrhea-like illnesses, including typhoid, cholera, and dysentery, represented the third largest cause of death in the nation.

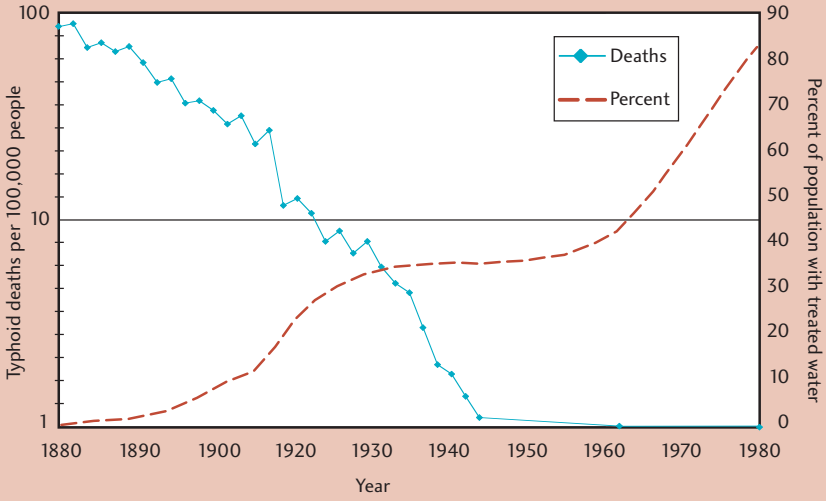
Then scientists identified the bacteria responsible for most diarrhea deaths (typhoid, cholera, and dysentery) and elucidated how these bacteria were transmitted to and among humans. Infected and diseased individuals shed large quantities of microbes in their feces, which flowed into and contaminated major water supplies. The contaminated water was then distributed untreated to communities, which used the water for drinking and other purposes. This created a continuous transmission cycle.

When treatment (filtration and chlorination) of drinking water was initiated to remove pathogens, the number of deaths due to diarrhea diseases dropped dramatically. Deaths due to typhoid fever were tracked throughout the early 20th century, as drinking water treatment was implemented across the country. Exhibit 4-3 shows the percent of the U.S. population that had treated water and the disease rate for typhoid fever from 1880 to 1980.

In this example, the outcome measure was death rates due to typhoid, which was used in conjunction with an environmental process (the number of people getting treated drinking water) to evaluate and promulgate the use of drinking water treatment across the U.S.

Drinking water treatment was one of the great public health success stories of the 20th century (NAE, 2000). It dramatically and significantly reduced death rates from waterborne disease, increasing

Exhibit 4-3: Percent of population with treated water versus typhoid deaths in the United States, 1880-1980



Source: Craun, C.G. *Waterborne Diseases in the United States.* 1986; Whipple, G.C. *Typhoid Fever - Its Causation. Transmission and Prevention.* 1908; Fox, K. National Risk Management Research Laboratory, personal communication, 2003.

life expectancy and reducing infant mortality. Today, public health is protected against new and emerging waterborne microbial contaminants by continual improvements to the drinking water treatment process and continual monitoring of waterborne diseases. Deaths due to cholera, typhoid, and dysentery are so rare in this country that they do not provide valuable information for evaluating the public health impacts of drinking water treatment. Instead, the number of cases of these diseases are tracked to some extent, although reporting is not federally required. Indicators for waterborne disease and other important diseases with actual or potential environmental origins are discussed in Section 4.3.

Case Study on Air Pollution

This case study illustrates how the association between deaths and peak air pollution concentrations was initially discovered by comparing mortality rates and air monitoring data. It also describes how basic research on the health effects of air pollution has helped to establish strong linkages between levels of certain air pollutants and human health effects. These associations have provided sufficient basis for establishing regulations to control the level of pollutants in air. The success of these environmental management efforts can be evaluated by monitoring levels of regulated pollutants in air. However, except for lead (the subject of the third case study below), there are as yet no biomonitoring or outcome indicators that can more directly measure reduced human exposure or outcome on a national level. Nevertheless, a number of potential outcome indicators are discussed that could be available in the future if systems can be set up to track relevant biomonitoring or outcome data with sufficient reliability and coverage at a national level.

Air pollution has been associated with several human health outcomes, including reported symptoms (nose and throat irritation), acute onset or exacerbation of existing disease (e.g., asthma, hospitalizations due to cardiovascular disease), and deaths. The impact of air pollution on health was underscored in London in December of 1952, when a slow-moving area of high pressure came to a halt over the city. Fog developed, and particulate and sulfur pollution began accumulating in the stagnating air mass. Smoke and sulfur dioxide concentrations built up over 3 days. Mortality records showed that deaths increased in a pattern very similar to that of the pollution measurements. (This is illustrated in Exhibit 4-4.) It was estimated that 4,000 extra deaths occurred over a 3- to 4-day period. This was the first quantitative air pollution exposure data with a link to an adverse health outcome (i.e., mortality).

While the London episode highlighted the hazard of extreme air pollution episodes, it was unclear whether health effects were associated with lower concentrations. By the 1970s, the association between respiratory disease and particulate and/or sulfur oxide air pollution had been well established (Dockery and Pope, 1997).

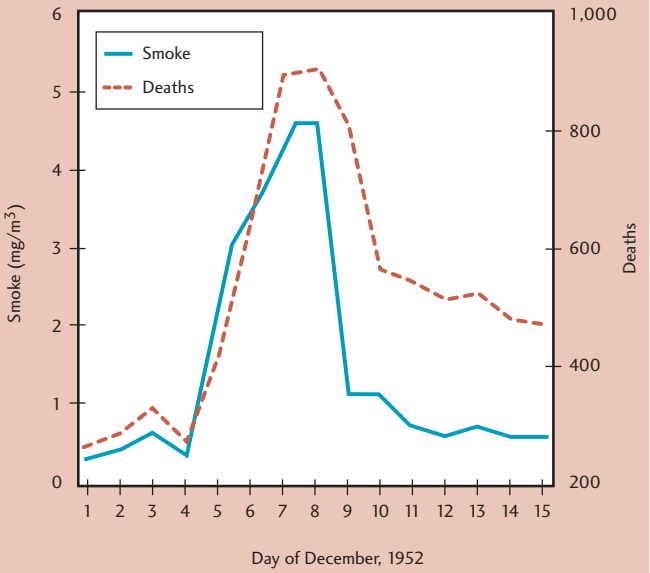
Clinical studies (controlled studies in healthy adult subjects) also provide information about the association between air pollutants and health effects. For example, these studies have demonstrated that ozone causes a number of functional, symptomatic, and inflammatory responses, which tend to increase with an increase in ozone exposure dose (EPA, 1996). Effects of ozone include:

- Decreased pulmonary function, characterized by changes in lung volumes and flow; changes in airway resistance and responsiveness; and respiratory symptoms, such as cough and pain on deep inspiration (EPA, 1996).
- An inflammatory response in the lungs (EPA, 1996).

Based on these types of associations from toxicological, epidemiological, and clinical studies, EPA has established National Ambient Air Quality Standards for six pollutants of concern: ozone, particulate matter, carbon monoxide, lead, nitrogen dioxide, and sulfur dioxide. These standards set limits to protect human health, including the health of "sensitive populations" such as asthmatics, children, and the elderly (EPA, 1999).

Improvements in measuring air pollution and health endpoints, together with advances in analytical techniques, have made it possible to begin to quantitatively evaluate the success of air pollution control measures—such as the National Ambient Air Quality Standards and associated regulations—to protect and improve public health. Though insufficient data were available at the time of this report to develop EPHIs for any criteria pollutants except lead, possible future EPHIs for air pollution include death due to respiratory and cardiovascular disease as well as increased hospital admissions for respiratory and cardiovascular disease.

Exhibit 4-4: London fog episode deaths, 1952



Source: Based on Dockery, D.W. and C.A. Pope. *Outdoor Air I: Particulates.* 1997.

Future EPHIs include:

- **Mortality.** In many countries including the U.S., particulate air pollution has been associated with increased daily mortality from heart and lung diseases (e.g., congestive heart disease, chronic obstructive lung disease). In addition, chronic exposure to air pollution has been linked with increased risk of premature mortality (EPA, April 2002).
- **Hospital admissions.** Hospitalization records are not widely available, and studies have been limited by their availability in communities around the U.S. Nevertheless, many studies have shown that increased admissions for cardiovascular and respiratory diseases are associated with increased pollutant concentrations.

Most recently, subtle changes in the cardiovascular system that can increase a person's risk of heart attack and bring about other cardiovascular effects have been identified as possible EPHIs.

Establishing EPHIs for air pollution and health effects, whether cardiovascular or pulmonary, is still challenged by limits in knowledge of how much air pollution contributes to the risk of both cardiovascular and respiratory disease. Research is still needed to better understand which components of air pollution (i.e., gases, metals, or organics) cause health effects; the extent to which they contribute to risk; and the extent to which other factors (e.g., genetics, lifestyle, age) contribute to risk. Given these limitations, no indicators are presented for any of the six criteria pollutants except lead. A case study on lead is presented below, with further discussion on lead as an indicator provided in Section 4.4.

Case Study on Lead

The third case study concerns lead, a toxic pollutant to which there is human exposure from many different sources. In the previous case studies, outcome indicators were an important key to establishing a linkage between a health effect and its cause. Understanding the cause enabled officials to take action to protect public health. In the case of lead, though it was a known toxin, exposure came from so many sources that it was difficult to know what actions at the national level would effectively reduce lead exposure. Once regulations to do so were put in place, biomonitoring data provided a way to evaluate the success of this environmental management effort in reducing exposure to lead in the U.S.

Lead is a neurotoxic metal that affects areas of the brain that regulate behavior and nerve cell development (NAP, 1993). Its adverse effects range from subtle responses to overt toxicity, depending on how much lead is taken into the body and the age and health status of the person (CDC, 1991).

Currently in the U.S., human exposure to lead may occur in several ways, as listed in Exhibit 4-5. For example:

- Homes built before 1978, commercial buildings, and steel structures may contain deteriorating lead-based paint, which creates lead-contaminated dust (EPA, 1996). An estimated 24 million housing units in the U.S. are at risk for containing some lead paint hazards (U.S. Department of Housing and Urban Development, 2000). Of these, 16 million homes with lead-based paint have children in residence who are younger than 6 years old.
- Other sources of lead exposure include lead-contaminated soil, dust, and drinking water; industrial emissions; and miscellaneous sources (CDC, 1991).

For many years, the largest source of lead in the U.S. environment came from leaded gasoline. Elemental lead was emitted in the exhaust and settled on the ground and in people's homes.

Most lead enters the body via ingestion and inhalation, after which it is absorbed by the bloodstream. Also, lead can cross the placenta, exposing the fetus to lead (EPA, 1996). In adults, most lead poisoning is associated with occupational exposures.

Infants, children, and fetuses are more vulnerable to the effects of lead because their blood-brain barrier is not fully developed (Nadakavukaren, 2000). In addition, ingested lead is more readily absorbed into a child's bloodstream. Children absorb 40 percent of ingested lead into their bloodstreams, while adults absorb only 10 percent. In children, three major organ systems are affected by lead: the nervous system (the brain), the kidney, and the blood-forming organs (NRC, 1993).

Exhibit 4-5: Sources of lead exposure in the United States

Lead-based paint	Homes (built before 1978) Commercial buildings Steel structures (bridges, water towers)
Lead-contaminated soil and dust	Industrial emissions Past leaded gasoline use Deteriorating lead-based paint
Lead-contaminated drinking water	Leaded plumbing solder (now banned)
Miscellaneous	Home hobbies - art, jewelry, fishing weights Use of pewter dishware Cosmetics, traditional medicines Parental occupations

Source: CDC. *Preventing Lead Poisoning in Young Children*. 1991.

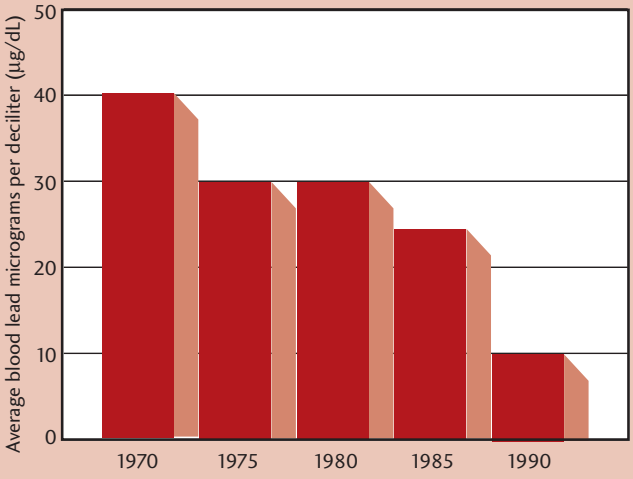
As awareness of the health effects of lead has increased, the CDC has lowered the level considered to be a human health hazard (Exhibit 4-6) (CDC, 1991). In 1970, a blood lead level of 40 micrograms per deciliter (µg/dL) or higher was considered a hazard. Today, 10 µg/dL or higher is considered a hazard (EPA, December 2000). Recent research suggests that blood lead levels less than 10 µg/dL may still produce subtle, subclinical health effects in children (Schmidt, 1999). In 1984, an estimated 6 million children and 400,000 fetuses were exposed to lead at levels that placed them at risk for adverse effects (NAP, 1993). Approximately 4.4 percent of all U.S. children in the 1990s had elevated blood lead levels (NCEH, 1998). As of 1998, an estimated 1 million U.S. children had blood lead levels above 10 µg/dL (NCEH, 1998).

Lead is one of the few pollutants for which biomonitoring and linkage data are sufficient to clearly evaluate environmental management efforts to reduce lead in the environment. The National Center for Health Statistics' National Health and Nutrition Examination Survey (NHANES), a national survey of the health status of the U.S. population, has determined blood lead levels for the U.S. population since the early 1970s. In the 1970s, lead poisoning occurred increasingly in children who did not live in dwellings with lead-based paint, suggesting that another source or sources of lead exposure were of even greater concern than lead paint. Research found that combustion of leaded gasoline was the primary source of lead in the environment. EPA promulgated two regulations:

- One required the availability of unleaded fuel for automobiles designed to meet federal emission standards (e.g., catalytic converters) (EPA, 1973).
- The second required a reduction of the lead content in leaded gasoline (EPA, 1986).

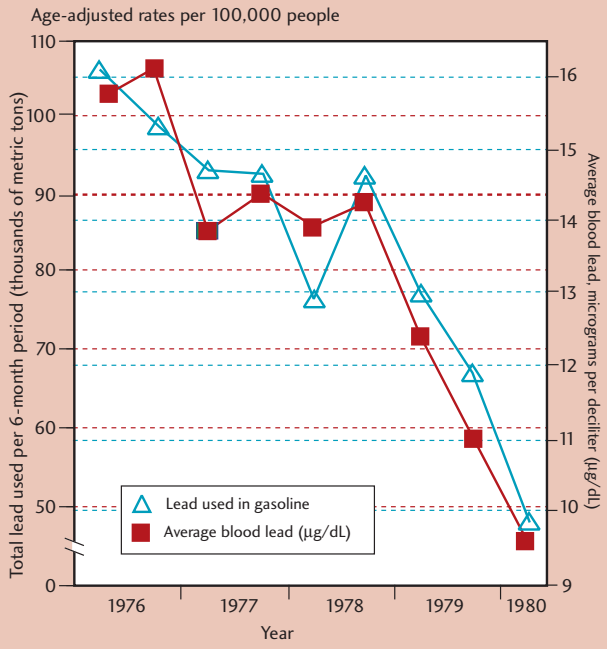
Over the next decade, peak outdoor-air lead concentrations decreased as a result of these controls. Exhibit 4-7 compares the amount of lead used in gasoline production and the average blood lead levels provided by the NHANES from 1976 to 1980. The NHANES survey found a similar decline in children's blood lead levels (Exhibit 4-8). In 1991, a report from the National Academy of Sciences predicted that declining ambient lead levels would reduce the average blood lead level to less than 15 µg/dL. By the late 1990s, the average blood lead level in the U.S. for children was 3 µg/dL (Schmidt, 1999). These data show a demonstrable effect between regulatory actions to control lead and human exposure.

Exhibit 4-6: Blood lead levels considered elevated by the Centers for Disease Control and Prevention and the Public Health Service, 1970-1990



Source: CDC. *Preventing Lead Poisoning in Young Children.* 1991.

Exhibit 4-7: Lead used in gasoline production and average National Health and Nutrition Examination Survey (NHANES) blood lead, United States, 1976-1980

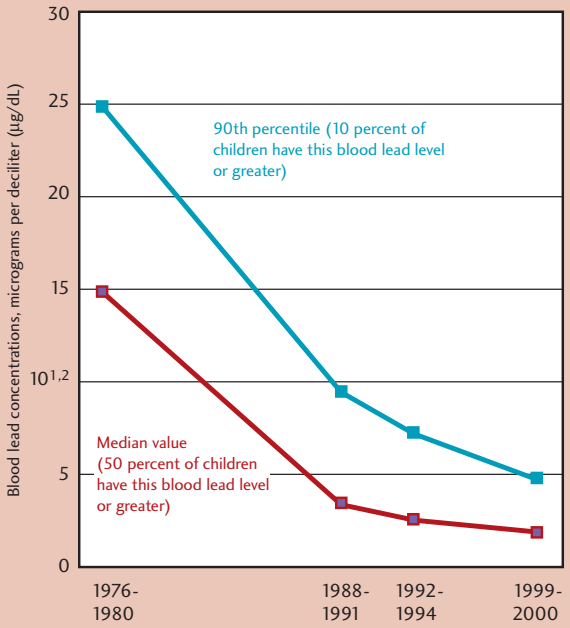


Source: National Research Council. *Measuring Lead Exposures in Infants, Children and Other Sensitive Subpopulations.* 1993.

Elucidating Other Linkages

For all three case studies, the linkage between exposure and disease is fairly strong. Subsequent sections of this chapter describe a number of areas of concern regarding the potential human health impacts of environmental exposure. The linkage in these areas ranges from strong to weak. For example, in some cases outcome indicators are available, but scientists are not yet sure how much of that outcome is contributed by environmental factors. In other cases, biomonitoring indicators are available, but scientists are not sure whether the presence of a contaminant in the body at the levels shown by the indicators causes adverse health effects. These areas are discussed in this chapter, despite relatively weak linkages, because the use of outcome and biomonitoring indicators is a developing area. Understanding of linkages will be strengthened over time as more research is conducted to develop environmental public health indicators and other data that reveal how pollutants contribute to disease.

Exhibit 4-8: Concentration of lead in blood of children age 5 and under, 1976-1980, 1988-1991, 1992-1994, 1999-2000



[1] 10 µg/dL of blood lead has been identified by CDC as elevated, which indicates the need for intervention. (CDC. *Preventing Lead Poisoning in Young Children.* 1991.)

[2] Recent research suggests that blood levels less than 10 µg/dL may still produce subtle, subclinical health effects in children. (Schmidt, C.W. *Poisoning Young Minds.* 1999.)

Source: U.S. Environmental Protection Agency. *America's Children and the Environment-Measures of Contaminants, Body Burdens, and Illnesses, Second Edition.* February 2003. Data from CDC, National Center for Health Statistics, National Health and Nutrition Examination Survey, 1976-2000.